Original Contributions

Risk Factors for Persistent Middle-Ear Effusions

Otitis Media, Catarrh, Cigarette Smoke Exposure, and Atopy

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• To ascertain risk factors for persistent middle-ear effusions (PMEE), we interviewed the parents of two groups of children. The first consisted of 76 children with PMEE who were admitted to the hospital for tympanostomy-tube insertion. The second, a control group, consisted of 76 children admitted for other types of surgery, who were matched for age, sex, season, and surgical ward. Nearly all (97%) of the children admitted for insertion of tympanostomy tubes had one or more episodes of suppurative otitis media. Only 59% of the control children had previous ear infections. Frequent ear infections sharply increased the risk for persistent effusions. Catarrh, household cigarette smoke exposure, and atopy also occurred more frequently in children with PMEE. The risk for middle-ear effusions was greatest when these three factors were all present. The avoidance of daily exposure to domestic tobacco smoke and, if atopic, of specific allergens should be included in the medical treatment of children with PMEE.

(*JAMA* 1983;249:1022-1025)

MIDDLE-EAR effusions are common in children, particularly after a suppurative middle-ear infection.[1,2] Most effusions resolve after several weeks, but some persist relentlessly,[3-7] causing hearing loss[8] and associated language, behavioral, and learning deficits.[9,10] Each year in the United States, an estimated 1 million operations take place in which tympanostomy tubes are inserted for persistent middle-ear effusions (PMEE).[11]

Several factors may affect the frequency of middle-ear disease: age,[2,12] sex,[12,13] season,[4] socioeconomic class,[12] exposure to other children,[2,14] catarrh,[2,5,6] positional feeding styles,[2,15] atopy,[16,17] and a family history of ear disease.[2] In this study, we examined the association of these factors with the persistence of middle-ear effusions.

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Reprints not available.

METHODS

The Research Committee and the Human Rights Committee at the Children's Orthopedic Hospital and Medical Center, Seattle, reviewed and approved these procedures. All parents gave informed consent before interview.

Case Selection

From June through October 1981, two general pediatric otolaryngologists performed 96 bilateral myringotomy and tympanostomy-tube insertions (BMT) for PMEE. Children were treated surgically if they had bilateral effusions (with pneumatic otomicroscopy and tympanometry) that did not resolve after eight or more weeks of medical therapy, and which produced a hearing loss of 25 dB or greater. These children were admitted to a short-stay ward at the Children's Orthopedic Hospital and Medical Center for surgery. Their parents were asked to participate in an interview about risk factors for ear disease. We interviewed 76 parents of the 96 patients with PMEE. Of the 96 patients' families, two were excluded because they did not speak English, and 18 could not be reached.

Control Selection

Twelve physicians (four general surgeons, one urologist, one ophthalmologist, two dental surgeons, and four cardiologists) allowed us to contact parents of their patients admitted during the same period to the same short-stay surgery ward. From this group of 202 children, control subjects were matched to PMEE cases by age (± 1 year), sex, and month of surgery. Ninety-five patients were matched initially, but 14 could not be contacted. Five interviews were excluded because of current middle-ear effusions or past ear surgery.

Clinical Characteristics of Cases and Control Subjects

Twenty-one patients with PMEE (27.6%) had previous bilateral tympanostomy-tube insertions (range, one to nine). Two patients with PMEE had Down's syndrome and two had cerebral palsy. In

the 76 control children, the reasons for admission were inguinal hernia repair (30), cardiac catheterization (17), biopsy or foreign-body removal (eight), umbilical, epigastric, or diaphragmatic hernia repair (six), orchiopexy (six), hydrocele repair (three), dental caries debridement (three), cystoscopy (one), esotropia repair (one), and proctoscopy (one). Down's syndrome occurred in only one control child who had cyanotic congenital heart disease. No other medical condition occurred more than once in either group.

Interview

Parents were interviewed within eight weeks of the scheduled surgery for the following information: (1) racial background, (2) family size, (3) health insurance status, (4) infant care and feeding practices, (5) household exposure to cigarette smoke, (6) frequency of suppurative otitis media (symptomatic ear infection treated with antibiotics), (7) frequency of catarrh (audible nasal breathing with rhinorrhea), (8) atopy (defined as one or more of the following disorders during the preceding 12 months: seasonal rhinitis [spring or summer sneezing, nasal itching, rhinorrhea, and nasal congestion]; asthma [recurrent wheezing, which improved with use of bronchodilators]; eczema [recurrent pruritic dermatitis, which improved with topical steroid therapy]), (9) family history of atopy, and (10) family history of significant middle-ear disease (six or more episodes of suppurative otitis media, or previous insertions of tympanostomy tubes).

Analysis

The likelihood of PMEE developing with a certain exposure was expressed as the relative risk and estimated using the Mantel-Haenszel method, standardizing for age (younger than 2 years, 2 years or older) and sex.[9] Ninety-five percent confidence intervals for each relative risk estimate were derived using the method of Miettinen.[10] For some factors, the relative risk changed with increasing exposure. We used an extension of the Mantel-Haenszel method[9] to test for a linear trend of changing relative risk.

RESULTS

Table 1 shows the frequency and relative risk for each of the interview variables. Patients and control subjects were similar in all socioeconomic and demographic categories. There were no significant differences in birth weight, early feeding patterns, the use of nighttime bottles, or daily exposure to other children. Exposure to two or more household cigarette smokers increased the risk for PMEE

Table 1.—Relative Risk of Persistent Middle-Ear Effusions (PMEE) According to Interview Variables

Characteristic	No. (%) of PMEE Cases (N=76)	No. (%) of Surgical Control Subjects (N=76)	Relative Risk*	95% Confidence Interval†
Demographic‡				
Sex				
M	46 (60.2)	46 (60.2)	...	...
F	31 (40.8)	31 (40.8)	...	...
Race				
White	66 (86.8)	66 (86.8)	1.0	...
Nonwhite	11 (14.5)	11 (14.5)	1.0	...
Household adults				
≥2	64 (84.2)	63 (82.9)	1.0	...
1	12 (15.8)	13 (17.1)	0.9	0.4-2.2
Siblings				
0	24 (31.6)	17 (22.4)	1.0	...
≥1	52 (68.4)	59 (77.6)	0.6	0.3-1.3
Health insurance				
Private	51 (67.1)	56 (73.7)	1.0	...
Nonprivate	25 (32.9)	20 (26.3)	1.4	0.6-2.8
Infant care‡				
Birth weight				
≥2,500	71 (93.4)	72 (94.7)	1.0	...
<2,500	5 (6.6)	4 (5.3)	1.3	0.3-5.0
First 6 mo				
Breast-fed only	23 (30.3)	21 (27.6)	1.0	...
Formula-fed only	33 (43.4)	28 (36.8)	1.1	0.5-2.7
Nighttime bottles (first 12 mo)				
Never used	47 (61.8)	52 (68.4)	1.0	...
≥5 nights per week	23 (30.3)	21 (27.7)	1.2	0.6-2.4
Daily exposure to other small children§				
None	37 (48.7)	36 (47.4)	1.0	...
At home only	14 (18.4)	10 (13.2)	1.4	0.5-3.8
At home and away	25 (32.9)	30 (39.4)	0.8	0.4-1.8
Irritant exposure				
Household cigarette smokers‖				
0	38 (50.0)	46 (60.2)	1.0	...
1	19 (25.0)	23 (30.3)	1.0	0.5-2.1
≥2	19 (25.0)	8 (10.5)	2.8	1.1-7.0
Household cigarette use,¶ packs per day				
None	38 (50.0)	46 (60.2)	1.0	...
0.1-0.9	11 (14.5)	7 (9.2)	1.9	0.7-5.3
1.0-1.9	13 (17.1)	14 (18.4)	1.1	0.5-2.6
2.0-2.9	7 (9.2)	8 (10.5)	1.0	0.3-3.1
≥3.0	7 (9.2)	2 (2.6)	4.1	0.8-19.2
Otitis media				
Suppurative otitis media,# episodes				
None	2 (2.6)	31 (40.8)	1.0	...
1-2	10 (13.2)	23 (30.3)	0.5	0.1-2.3
3-5	12 (15.8)	19 (25.0)	0.7	0.2-2.8
>5	52 (68.4)	3 (3.9)	166.7	46.4-667
Age at first otitis, mo				
≥6	38 (48.7)	23 (73.3)	1.0	...
<6	38 (51.3)	12 (26.7)	3.0	1.2-7.4
Family history of middle-ear disease				
Absent	42 (55.3)	53 (69.7)	1.0	...
Present	34 (44.7)	23 (30.3)	1.9	0.9-3.8
Nasal congestion (see text for definition)				
Frequency of symptoms,** days monthly				
None	31 (40.8)	57 (75.0)	1.0	...
<5	10 (13.2)	6 (7.9)	3.0	1.0-8.8
5-15	15 (19.7)	6 (7.9)	4.0	1.7-12.0
>15	20 (26.3)	7 (9.2)	5.3	2.0-12.5
Atopic disease (see text for definition)				
Frequency of atopic symptoms,†† days monthly				
None	54 (71.0)	66 (86.8)	1.0	...
1-15	7 (9.2)	6 (7.9)	1.4	0.4-4.8
>15	15 (19.8)	5 (6.6)	3.7	1.3-10.6

Table 1.—Relative Risk of Persistent Middle-Ear Effusions (PMEE) According to Interview Variables (cont)

Characteristic	No. (%) of PMEE Cases (N=76)	No. (%) of Surgical Control Subjects (N=76)	Relative Risk*	95% Confidence Interval†
Atopic disease (cont)				
Family history of atopic disease				
Absent	35 (46.0)	36 (47.4)	1.0	...
Present	41 (54.0)	40 (52.6)	1.1	0.6-2.0

*Standardized for age and sex by the method of Mantel and Haenszel.[30]
†Approximate limits, calculated by the method of Miettinen.[31]
‡Mean age ± SD was 3.52 ± 2.7 years for the PMEE cases and 3.37 ± 2.6 years for control subjects. Mean birth weight ± SD was 3,349 ± 681 g for PMEE cases and 3,335 ± 568 g for control subjects.
§Test for linear trend,[30] comparing strata of increasing exposure ($P=.48$).
‖Test for linear trend[30] ($P<.001$).
¶Test for linear trend[30] ($P<.05$).

Table 2.—Combined Effects of Risk Factors for Persistent Middle-Ear Effusions (PMEE)

Attributes	No. (%) of PMEE Cases (N=76)	No. (%) of Surgical Control Subjects (N=76)	Relative Risk*†	95% Confidence Interval‡
None	16 (21.0)	31 (40.8)	1.0	...
Only 1 factor	29 (38.8)	33 (43.4)	1.8	0.7-3.6
Congestion (>1 day a month)	14 (18.4)	7 (9.2)	3.9	1.3-11.3
Smoking (>0.5 packs per day)	13 (17.1)	22 (28.9)	1.1	0.6-2.0
Atopy (>1 day a month)	1 (1.3)	4 (5.3)	0.6	0.06-4.6
2 factors combined	19 (25.0)	8 (10.5)	4.8	1.7-12.5
Smoking and congestion	11 (14.5)	5 (6.6)	4.3	1.3-13.9
Smoking and atopy	1 (1.3)	0 (0.0)	...	...
Congestion and atopy	7 (9.2)	3 (3.9)	4.6	1.1-18.7
All 3 combined	13 (17.2)	4 (5.3)	6.3	1.9-21.1

*Standardized for age and sex by the method of Mantel and Haenszel.[30]
†Test for linear trend comparing none, one, two, and three factors ($P<.001$).[30]
‡Approximate limits, calculated by the method of Miettinen.[31]

nearly threefold. With household exposure to smoke from more than three packs of cigarettes per day, the risk increased fourfold.

Nearly all of the patients with PMEE had one or more previous episodes of suppurative otitis media. A significant trend of increasing relative risk occurred with increasing frequency of otitis media. When the first episode of otitis media occurred at younger than 6 months of age, there was an apparent threefold risk for PMEE. However, if the age at the first episode of otitis was standardized for the total number of episodes, the relative risk was only 1.6 (95% confidence interval, 0.6 to 4.5). Thus, early otitis media may increase the risk for more frequent episodes of suppurative otitis, but of itself does not significantly increase the risk for PMEE. A family history of ear disease increased the risk less than twofold, but despite this modest elevation, families with three or more affected members occurred only in the PMEE group.

Nasal congestion occurred more often, and was more persistent, in children with PMEE. With more persistent catarrh the risk increased from threefold to fivefold. Atopic diseases occurred twice as often in children with PMEE. In those who required repeated tympanostomy-tube insertion, ten (48%) of 21 had atopic disease. The risk for PMEE increased nearly fourfold in children with persistent atopic symptoms. A family history of atopic disease did not increase the risk for PMEE.

Table 2 shows the combined effects of nasal congestion, cigarette smoke exposure, and atopy. Nasal congestion alone elevated the risk nearly fourfold. When cigarette smoke exposure or atopy was added to nasal congestion, the risk increased. Children with all three factors were more than six times as likely to manifest PMEE.

COMMENT

Suppurative otitis media, catarrh, household cigarette-smoke exposure, and atopy are important risk factors for the development of PMEE. The risk increases with more long-term exposure. Several clinical and laboratory studies would substantiate the importance of these factors. Recurrent infections can damage ciliary function and cause metaplastic changes in middle-ear mucous glands.[2] The altered mucosa secretes a thick, gluelike fluid, which is more likely to persist for long periods. Catarrh, which occurs more commonly in children with abnormal middle-ear pressures,[3,4,5] may reflect repeated nasal infections, nasal irritant reactions, or nasal allergy. Each of these conditions could cause mucosal edema, hypersecretion, and abnormal ciliary function, which then results in obstruction or "dysfunction" of the eustachian tubes. Passive childhood cigarette smoke exposure increases the frequency of nonallergic respiratory symptoms[26,27] and may aggravate respiratory allergies.[28] In heavily exposed children, catarrh from infection or allergies could become more persistent. In children with atopic disease, allergic rhinitis is the likely cause of their increased risk of middle-ear effusions. Recent studies in patients with allergies have shown that nasal challenges with specific antigens can produce sustained abnormalities of eustachian tube function.[29,30]

Recurrent otitis media, nasal catarrh, cigarette smoke exposure, and nasal allergies chronically inflame the nasal and middle-ear cavities, causing persistent eustachian tube dysfunction. Middle-ear effusions will clear less readily in heavily exposed children, which may eventually necessitate surgical drainage and insertion of tympanostomy tubes. For these children, a medical treatment plan should include the elimination of tobacco smoke from the domestic environment and, if atopic disease is present, the control of specific environmental allergens.

This study was supported by a grant from the Children's Orthopedic Hospital and Medical Center Research Fund R2672, and by Associated Scientists to Help Minimize Asthma (ASTHMA) Inc, Seattle.

James Donaldson, MD, Barb Anderson, RN, and Donnie Lavery provided help in contacting patients. The physicians, surgeons, and support staff of the Children's Orthopedic Hospital and Medical Center, Seattle, provided assistance in contacting control subjects. Nancy Kraemer assisted in preparation of the manuscript.

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